

Carroll (A. L.)

With the author's regards

REMARKS INTRODUCTORY TO A DISCUSSION

ON

ACUTE DIFFUSE PERITONITIS

By A. L. CARROLL, M. D.

*[Reprinted from the Transactions of the New York State Medical
Association for 1891]*



CONCORD, N. H.

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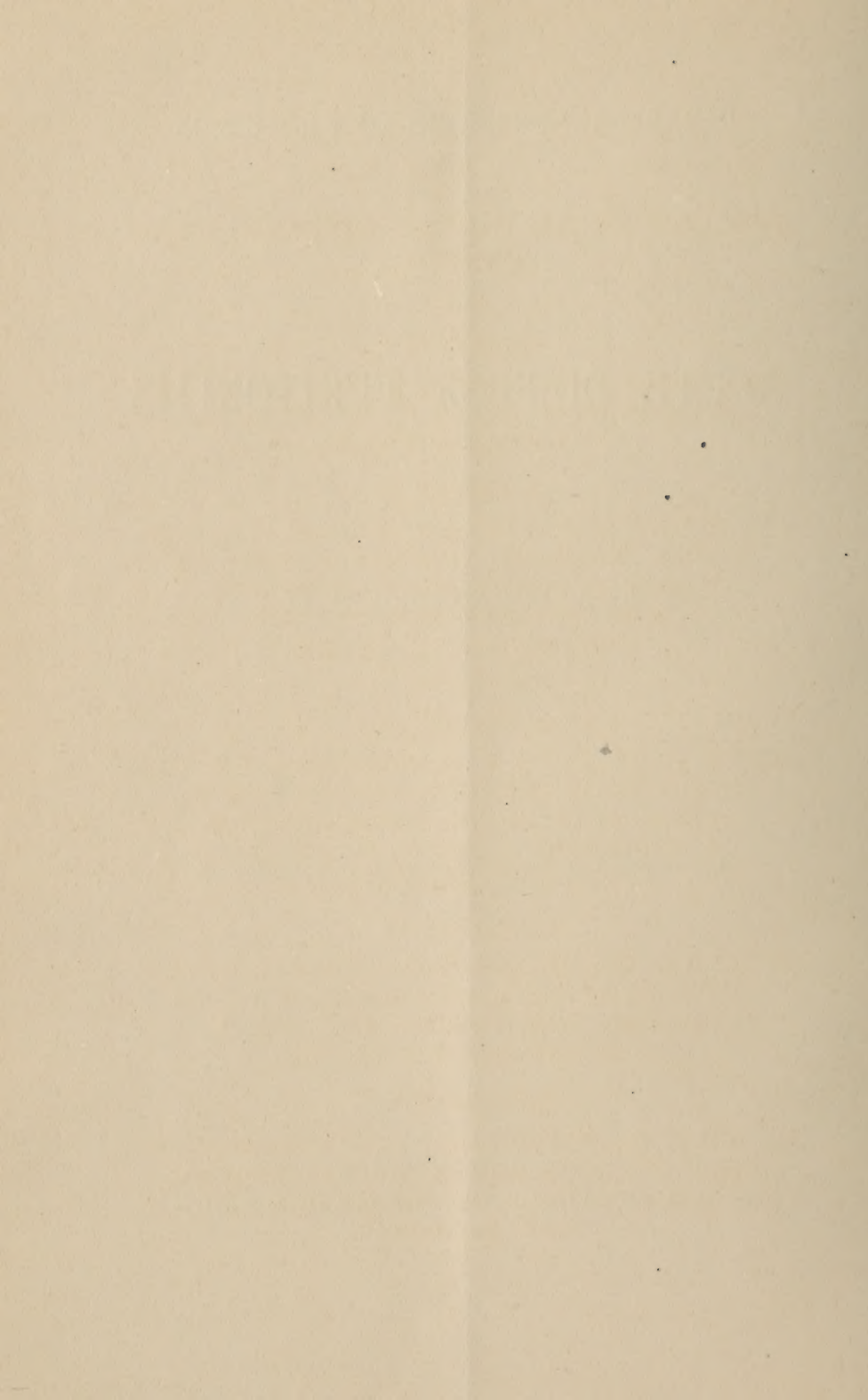
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DISCUSSION ON ACUTE DIFFUSE PERITONITIS.

QUESTION I.

WHAT DOES THE NORMAL HISTOLOGY OF THE PERITONAEUM TEACH US OF ITS PHYSIOLOGICAL PURPOSE?

QUESTION II.

DO PATHOLOGICAL AND CLINICAL CONSIDERATIONS WARRANT THE SEPARATION OF CASES OF ACUTE DIFFUSE PERITONITIS INTO DIFFERENT CLASSES, SOME INFECTIVE, OTHERS NOT SO?

QUESTION III.

WHAT PART DO MICRO-ORGANISMS, OR THE PRODUCTS OF FERMENTATION CAUSED BY THEM, PLAY IN THE PATHOGENY OF DIFFERENT FORMS OF ACUTE DIFFUSE PERITONITIS?

QUESTION IV.

WHAT ARE THE MEANS OF DIFFERENTIAL DIAGNOSIS BETWEEN VARIOUS FORMS OF ACUTE DIFFUSE PERITONITIS, AND WHAT THE PROGNOSIS IN EACH?

QUESTION V.

IN WHAT RESPECTS ARE THE THERAPEUTIC INDICATIONS IN ACUTE DIFFUSE PERITONITIS MODIFIED BY THE AETIOLOGICAL FACTORS?

- (a) *From wounds, contusions, rupture of bladder or other viscus, or of abscess or aneurism; perforation; intussusception or strangulation; typhlitis; caries of bone; gallstones; infection in course of lymphangitis.*
- (b) *From fecal impaction; extension from enteritis; hepatitis, or disease of other abdominal viscera; in dysentery, or typhoid ulceration without perforation; incident to paludal or exanthematic fevers, to albuminuria, scurvy, cardiac disease, rheumatism; from exposure to cold and damp; from decomposition of intestinal contents.*
- (c) *In women: from imprudence or exposure during menstruation; extension from pelvic peritonitis, peri-uterine cellulitis, metritis, salpingitis, ovaritis, etc.; from gonorrhoeal infection; from abortion; puerperal, extending from metritis, or by septicaemia; sequent to abdominal section.*
- (d) *In children: from umbilical phlebitis; intussusception; worms, etc.*

REMARKS INTRODUCTORY TO THE DISCUSSION.

By ALFRED L. CARROLL, M. D., of New York County.

October 28, 1891.

Question I. What does the normal histology of the peritoneum teach us of its physiological purpose?

The first question on our programme might have been not inaptly preceded by the inquiry, What is a "serous membrane"? and, simple as the query sounds, there would be diversity of opinion in the answers to it; for neither in embryological origin, in apparent physiological use, nor in reaction to morbid processes, is there correspondence between all the textures which are grouped under this generic title. The epiblastic ocular chambers and aural labyrinths still hold their places in our text-books, notwithstanding that the

normal purpose of their contained fluids is rather to maintain separation of surfaces than to relieve friction. So, too, the single-layered, non-vascular arachnoid bears a very doubtful relationship to the thoracic and abdominal membranes in either its histology, or its normal or morbid behaviour. And when we find an instructor in a prominent medical school proclaiming that "The nasal mucous membrane is a serous membrane," interrogation is paralysed, and all hope of definition is abandoned.

The similarity of the original structure of the pericardium, pleura, and peritoneum—with its offshoot, the tunica vaginalis testis—is taught by the embryology of the pleuro-peritoneal cavity, and confirmed by the fact that in sundry cases of arrested development two or more of these remain in inter-communication; and if in perfected evolution any differencing of such structure occur, it may be assumed to entail a difference of function. Of course, the consideration of structure here intended is not confined to the old anatomical description of a "serous membrane" as composed of a stratum of squamous cells superposed upon a "structureless basement membrane," but must include all tissues that are tributary to the purpose served.

All three of these sacs consist mainly of interwoven connective tissue bundles and elastic fibres, lined by endothelial cells, and of a "subserous" connective tissue layer of varying density, wherein are contained such blood-vessels, nerves, and lymphatics as they may respectively possess; in all, the parietal is thicker than the visceral layer, which is more or less intimately associated with the invested viscera; all are moistened by fluids, of the nature and mode of production of which the present state of our ignorance leaves us in troublesome uncertainty. Once a similitude to blood-serum gave the membranes their name; later, a nearer analogy to blood-plasma was discerned; and in both instances the physical phenomena of osmosis were invoked in explanation, the synovial sacs being drafted into a separate regiment because the viscidify given to their contents by "synovine" argued a

secreting power of their endothelium—as if any living cells could refrain from modifying the character of the blood-constituents with which they have to deal; still later, it has been asserted that the osmosed or secermed fluid is identical with lymph, with the somewhat important omission of the lymph-corpuscles, and our “serous membranes” are rebaptised as lymphatic sacs.

But beyond their general conformity, each has certain peculiarities: The external surface of the pericardium and the costal aspect of the pleura are covered by a tougher fibrous lamella than is found in the parietal peritoneum. The vascularity of the pericardium is shown, not only by normal anatomy, but by its “red velvet” appearance when acutely inflamed. The pleura, though in less degree, is well supplied with blood, especially in its pulmonary layer. Both have numerous nerve-filaments. Both seem—like the synoviae and bursae—to subserve in health principally the office of lubricating apposed surfaces in nearly constant motion.

Turning now to the peritoneum, the first difference that strikes the eye is in its mechanical distribution. While the only noteworthy pleural fold is the ligamentum latum pulmonis, a large part of the peritoneum is devoted to suspensory uses, or forms extravagant pouches: The ligaments of the liver, spleen, uterus; the mesenteries, the quadruplication of the greater omentum, interposing an apron between the parietal and visceral layers, and inclosing a separate cavity of its own; these and minor foldings and pockets give an enormous extent of endothelium, the stomata of which lead in most places to a loosely-meshed connective tissue, rich in lymph-spaces and lymphatic vessels. Its blood-supply is scanty, its nerves few, and these chiefly in the omenta and mesenteries, the latter bearing the intestinal lymphatics and mesenteric glands. Many sub-serous lymphatics of the liver, especially those surrounding the gall-bladder, conduct to glands in the gastro-hepatic omentum; others, originating in the muscular coats of the hollow viscera, have been traced in the sub-peritoneal tissue. In short, the peritoneum offers a

far more elaborate absorbent apparatus than any other of the membranous sacs, and to this its mere lubricating office is apparently subordinate. Experiments have demonstrated the rapidity with which injected liquids are imbibed, and the possibility of maintaining nutrition by this means.

In women, an additional channel of invasion exists in the Fallopian tubes, beside the communication of the lymphatics of the broad ligament with those of the uterine body, and the increase of the latter during pregnancy.

Question II. Do pathological and clinical considerations warrant the separation of cases of acute diffuse peritonitis into different classes, some infective, others not so?

In a vast majority of cases of peritonitis the morbid process begins in the sub-peritoneal connective tissue of the visceral layer, the virtual continuity of which, with the connective tissue stroma of the invested organs, and its lymph-spaces derivative from them, offer facilities for either direct extension of common inflammation or transference of septic products, thus affording ground for a distinction between at least two classes of pathic conditions secondary to local visceral disease. But in addition to these we have cases in which the inflammation is excited by mechanical or toxic agents operating immediately upon the peritoneum; others, wherein it takes rise from lymphangitis or general septicaemia; still others, such as those sometimes called "idiopathic," of which the pathogeny is not always discoverable. Here, again, the grouping of infective and uninfected is pathologically and clinically manifest; but we know—without knowing exactly why—that, especially in what may be termed the surgical category, a case may occasionally transfer itself from the milder to the graver sort.

The effusion in peritonitis, as in pleuritis, may be plastic, sero-fibrinous, purulent, or ichorous; and the character of it seems to depend more upon the aetic factor than upon the intensity of the inflammatory process, of the real nature of

which we know little or nothing. A mere adhesive film may be produced where vascular injection and subserous infiltration are marked; a profuse sero-fibrinous fluid may be poured out where at most an opalescence of the membrane is discernible; while in septic peritonitis, particularly in puerperae, Rokitsansky first called attention to the amount of toxic, disorganised exudates with hardly perceptible reddening or vascularity.

The plastic form is oftenest seen in peritonitis by extension of aseptic inflammation from subjacent organs, or from "contusion"—which, in view of the now notorious tolerance of pretty rough handling by the peritonaeum, I am inclined to regard in most instances as nearly the same thing. The exudate may become organised into bands or more or less adherent layers of varying extent, or create papillary or dendritic roughenings of the surface; or its redundant cell elements may degenerate into pus, giving rise in some examples to a secondary infective process, in others remaining "laudable," to undergo fatty change and be absorbed, or, losing their liquid part, to be calcified.

The more abundant sero-fibrinous effusions, frequently observed in cases arising from exposure to cold and damp, from faecal impaction, from strangulated hernia, etc., may, under favourable conditions and judicious treatment, be rapidly and completely absorbed, or, as in the preceding class, may result in adhesions, or even in suppuration.

Purulent peritonitis is common from mechanical or chemical irritants, or from putrid infection; as in perforation, rupture of the urinary bladder, operation for imperforate hymen after puberty, septic or suppurative pelvic disease in women, etc., as well as from the above mentioned degeneration of more innocent exudates; and in children the proclivity to pus formation in peritonitis is greater than in after life.

I shall not advert to the still waging controversy concerning the origin of pus and other cell elements in these exudates, farther than to say that the exclusive ascription of all of them to the migration of leucocytes does not seem to me

to be sufficiently justified. The leucocytes in their origin are embryonic, connective tissue cells, proceeding apparently from the peripheral juncture of the two layers of the meso-blast. Even in mature life, according to our present uncertain doctrine, they are newly formed in the bone-marrow, spleen, and lymphatic glands, and probably in other unknown organic laboratories. All protoplasm, unhindered by a rigid limiting envelope, possesses, as far as comparative biology teaches us, amoeboid movement; and I have yet to be convinced that "wandering" connective tissue corpuscles are necessarily lost leucocytes which have strayed from a distant intra-vascular home, or that new growth of connective tissue is essentially dependent on leucocytic emigration. Although pus corpuscles are unquestionably enfeebled, we have no positive authority for declaring that they are "dead leucocytes." In the case of peritonitis, particularly, the enlargement of the endothelia, the active subdivision of their nuclei, and the appearance of multitudes of new cells in their place, are noted by careful investigators. In fine, not much accurate progress has been made since Waller, nearly half a century ago, showed the impossibility of a scientific discrimination between the leucocyte, the pus corpuscle, the "granulation cell," and, I would add, the primitive connective tissue corpuscle. It will also be noticed that I am not prepared to accept the "microbic" origin of all inflammation.

Question III. What part do micro-organisms, or the products of fermentation caused by them, play in the pathogeny of different forms of acute diffuse peritonitis?

As bio-chemistry has trodden on the heels of bacteriology, the intrinsic and immediate maleficence of particular "pathogenic" microphytes has been discussed with increasing skepticism, and we have learned that chemical products of their reaction upon an appropriate environment, or of their own decay, are the real morbid agents. Furthermore, there is reason to believe that the nature of the material on which

they act is an essential factor in the property of the product, and Vaughan's experimental research confirms the opinion entertained by several observers, that microphytes differing in form and in susceptibility to staining reagents may cause the development of poisons similar, if not identical, in effect. Beside the alkaloidal ptomaines or "toxines," proteid substances or "toxalbumins" have also been isolated and their virulence demonstrated. According to Büchner, however, very few of these products are capable of exciting suppuration, which he attributes to what he calls "bacteria-proteins," arising from the plasma of the dying or decadent microphytes themselves, and chemically resembling the vegetable caseins.

We have learned, too, that without bacterial intervention toxic compounds may be formed within the body; that leucomaines are not only producible by perverted action of the living animal cells, but result from many of the physiological processes of metabolism; that the very peptones of normal digestion are poisonous if introduced directly into the circulation; that the misbehaviour of our own tissue elements, or the defective elimination of the waste-products of their activity, may do nearly as much mischief as an exogenous bacterial virus. Diminished resistance or degradation of the cells, moreover, facilitates microbial invasion and multiplication, and converts the patient, so to speak, into a "culture-medium."

Most of the infective forms of peritonitis are probably occasioned by the products of bacterial fermentation, and these products may apparently be elaborated by various saprophytic micro-organisms. Examples are found in peritonitis from dirty, penetrating wounds, from perforation or laceration permitting the ingress of faecal or purulent matter, from entrance of decomposing material from the uterus and Fallopian tubes, from general septicaemia, etc. But there are cases presenting somewhat similar phenomena which are perhaps attributable to endosomatic poisons, generated or accumulated in the disordered tissues. In intussusception or strangulation of any kind, in severe bruising without exter-

nal wound or laceration, where circulatory disturbance leads to perversion of nutrition or to molecular necrosis, the conditions exist for incidental noxious synthesis.

The intense and rapidly diffused inflammation excited in numerous instances by intra-peritoneal effusion of urine from rupture of the bladder through its serous investment, is not easily to be classified. On the one hand, normal urine, unexposed to air, is aseptic; on the other, none of its ordinary ingredients is known to be violently irritant; in fact, it is the ancient mariner's favourite collyrium for conjunctivitis. Even if the conversion of its urea into ammonium carbonate were chemically imaginable in these circumstances, the latter salt, in much stronger solution than is possible in urine, has been tested upon animals by intravenous injection and otherwise, with only the temporary manifestation of its tetanising influence. There is some evidence that urine may be absorbed by the peritoneum, and more that its prolonged presence in the serous sac does not always induce inflammation. Whether a septic ferment is imported, or a proteid or leucomainal poison concerned in such cases, is a problem for future bio-chemistry to solve.

Question IV. What are the means of differential diagnosis between various forms of acute diffuse peritonitis; and what the prognosis in each?

If we discard the too long misused adjective "idiopathic," as applied to peritonitis, it is evident that diagnostic inquiry must often take a wide range to discover the originating disorder; and in some instances,—such as those arising from incarceration, non-perforative implication in typhoid fever, nephritis, etc.,—the prognosis depends rather on the primary disease than on the peritoneal involvement. Even where peritonitis, per se, constitutes the principal peril to life, prognosis—as in other pathic conditions—is a variable term, changing with every advance of therapeutic art.

It is scarcely needful, in addressing an audience of experi-

enced physicians, to remark that the early diagnosis of peritonitis is not always as facile as some writers represent it. Initial rigour and rapid rise of temperature are frequently absent, and when they do occur, are commonest in cases caused by septicaemia, or by exposure to cold—cases with totally opposite prognosis. Indeed, there are instances in which the whole course of the disease is nearly or quite afebrile, and others wherein the temperature may be subnormal. The pain and tenderness, ordinarily so characteristic, may, in rare examples, be trifling, as if the peritoneum retained its natural insensitiveness despite the inflammatory provocation. Nausea and vomiting are seldom inceptive symptoms, and may exist in other disorders—as in enteritis or intestinal occlusion—which are among the not infrequent causes of peritonitis; and constipation falls in the same category. Tympanites is an indication that inflammation and infiltration have already made much progress, and, in muscular patients, may not appear until near the end. Fluctuation is only discoverable where an abundant liquid exudation has taken place. Oliguria and dysuria are neither peculiar to peritonitis, nor are they so marked in this when exudation is slight and vomiting scanty or absent. An expert diagnostician may now and then find himself with little more than the physiognomy and decubitus of the patient to guide his first opinion. Granting that such cases are exceptional, it is nevertheless important to bear in mind their existence.

Still more difficult is it sometimes to determine when diffuse peritonitis supervenes upon preëxisting maladies or traumatism, where the symptoms of the primary lesion simulate or mask those of the secondary affection. As a rule (with not a few exceptions), the marked increase and unremitting character of pain, the change from restlessness to immobility and relaxation of the abdominal muscles, the rise of temperature, the quickening of the pulse, and the onset of vomiting (if not previously present), will furnish the earliest warnings. In many surgical cases, fortunately, this diffi-

culty of diagnosis is of less moment, because the lesion itself justifies operative interference, which thus becomes prophylactic.

The rapid course of traumatic peritonitis renders its prognosis at all times very grave—worst if there be wound of the intestine or other abdominal viscus. In scarce instances, limiting adhesions may form a barrier of safety; but in others the exudation speedily assumes a sero-purulent or ichorous character, and life is soon extinct. Here, surgery should be occurrent against the coming ill, not awaiting its arrival.

Contusion in minor degree oftenest, probably, primarily affects an invested viscus, leading thence to a local peritonitis, which may become diffuse, as in other examples of extension from simple visceral inflammation. Sharp pain and tenderness do not appear until a considerable time—several days, even—after the accident, beginning at the injured point and gradually spreading. The exudate is ordinarily plastic or sero-fibrinous; the prognosis not unfavourable, if the visceral inflammation be resolved. If severer contusion cause lesion of the peritoneum itself, the onset of inflammation is more sudden and the extension more rapid; the prognosis—especially if any hæmorrhage ensue—less hopeful. If crushing violence produce actual rupture of viscera—as of the stomach, intestine, liver, kidney, bladder—with effusion of blood, faeces, or other contents, the diagnosis of the fact, though not always of the seat of the solution of continuity, is generally self-evident; but it is well to remember the cases recorded from time to time since Bransby Cooper's day, in which the severest lesions of this kind were not immediately followed by any characteristic symptoms. The supervention of fatal peritonitis upon such hurts was almost inevitable before modern surgery with disinfectant methods gained courage to attack the causes; and even now the chances of recovery are small. The diagnosis of peritonitis in the very few instances arising from rupture of the spleen or of an aneurysm has hitherto been made post mortem, when prognosis had lost its interest as a question for practical consideration. Abscess usually can,

and should, be detected and relieved before its rupture into the peritoneum.

Ulcerative perforation of the peritoneum commonly gives rise to a quickly diffused inflammation, following collapse induced by the accident, the symptoms being similar to those produced by rupture. If the perforation be small, and the intrusion of the contents of the digestive tract very scanty or gradual, limiting adhesions may form, or diffuse peritonitis may occur with few diagnostic indications of its advent. In most of these cases the determination of the exciting cause and of its situation must depend chiefly upon the recognition of preëxisting disease by the physician who has had previous charge of the patient; and every physician should be aware of the possible surgical outcome of a medical malady, and of the fatal mischief of delay when this possibility becomes an actuality.

Gastric ulcer occasionally, though seldom, may escape detection until perforation brings on a rapidly mortal peritonitis. Intestinal ulceration, on the other hand, is always difficult to diagnosticate in its earlier stages, and especially so when arising from uncommon causes. In malarial fevers accompanied by intestinal catarrh, ulceration or sloughing of the colonic glands, or even of Peyer's patches, may proceed to perforation, and the same result may take place in dysentery if the inflammation advance to suppurative disintegration of the muscular coat, the danger here, however, being foreshadowed by the markedly purulent character of the stools. Typhlitis with caecal impaction—ordinarily of favourable prognosis—may possibly lead to perforative ulceration, as may also other mechanical obstructions. Perforation occurring as an incident of enteric fever hardly pertains to our discussion, since it is virtually irremediable by any present resources of our art.

The commonest source of perforative peritonitis is inflammation of that degrading reminder of our evolution from a herbivorous ancestry, the vermiform appendix, which, beside its direct attacks on the serous membrane, is at the bottom

of about ninety per centum of the cases of perityphlitis. And the discovery of this condition is not always easy, for its course may be insidious, with normal temperature, little pain, absence of caecal tumefaction, latency of all characteristic symptoms until the approach of death. I have seen a patient whose only apparent discomfort was obstinate constipation, without abdominal distention, with so little tenderness that he bore without inconvenience sufficiently deep palpation to detect slight thickening in the inguinal region and obscure fluctuation below, and yet incision revealed intense diffuse peritonitis with foul purulent exudation, the appendix perforated, and a faecal concretion extruded.

Supposing that in cases of sudden onset, unheralded by prior ailment, the medical attendant has followed the stereotyped injunction to examine the ordinary hernial openings, the differential diagnosis of internal strangulations or occlusions is generally impossible except by exploratory section. Intussusception—commonly ileo-caecal in youth—may be surmised from the local and often remitting nature of the initial pain and the accompanying dysenteric stools, and verified by palpation; but in the rarer instances where invagination occurs higher in the small intestine, the sense of touch is often baffled, and even if tumor be felt its cause cannot be discriminated from other sources of occlusion. In all such cases purulent, and sometimes gangrenous, peritonitis is likely to result, and more than in other classes is prognosis linked with prompt treatment.

Faecal impaction may induce peritonitis attended with little, if any, rise of temperature, tympanites, or pain, and therefore apt to be overlooked in its earlier stage. In the majority of instances, however, it occurs in the caecal region, and an intermediate typhlitis is discoverable. If the cause be removed, recovery is likely. In cases ensuing upon renal or cardiac lesions, or severe constitutional disorders, the issue is dependent chiefly on the originating disease.

In women, the prophetic discrimination between infective and uninfected inflammation of the peritonaeum is more

important than in men, and often harder to achieve. At best, extension from pelvic peritonitis is of graver presage than that arising from simple affections of abdominal viscera, except, perhaps, in the comparatively benign form which sometimes follows exposure during menstruation, and which has been theoretically ascribed to the intervention of perimetritis. But since peritonitis, like pleuritis, is occasionally induced in men by subjection to cold and damp, it is not always necessary to imagine a distinctively sexual factor otherwise than as increasing susceptibility, save where the menstrual flow is intentionally arrested. Salpingitis as a cause of peritonitis has assumed great prominence of late, and is undoubtedly the intermediary in many infective examples, the fatality of which has been much reduced by gynaecological progress. The frequency of gonorrhoeal invasion through this channel, however, is unreasonably overrated, even by some of those who stop short of Noeggerath's extreme views. We know that the unmistakably specific vaginitis very seldom spreads above the os internum. Daily experience shows that the reliquial discharge of gleet—and, *a fortiori*, any casual serous or mucous secretion long afterward—fails to convey specific virus; and the "gonococcus" (or its twin Dromio) is not confined to gonorrhoeal pus. Sepsis from a retained portion of the ovum, or metritis from mechanical injury, may lead to peritonitis after abortion, as in the puerpera there may be different pathogenic grades, of widely varying significance, from simple metritis to putrid absorption, or to the mysteriously overwhelming septicaemia which may kill before an exudate has formed.

During infancy, the prognosis of peritonitis, from whatever cause arising, is serious in inverse ratio to age. When consequent to infection from umbilical phlebitis or gangrene, within the first few weeks of life, a fatal termination is inevitable. This dangerous period past, the exciting factors differ little from those in adults, but the peril of the resulting peritonitis itself is far greater.

In these prognostic hints the immediate outcome of acute

inflammation only has been considered, the occasional transition into a chronic form not concerning our purpose.

Question V. In what respects are the therapeutic indications in acute diffuse peritonitis modified by the aetiological factors?

It can never be too often reiterated that, while the naming of a morbid condition is a provisional necessity for registration, the given title very rarely conveys a therapeutic indication; and in the case now under deliberation it would be small loss if the word "peritonitis" were relegated to the limbo whereto are already consigned the terms "dropsy" and "paralysis," the latter of which, by the bye, has been misconconnected—nosographically, if not etymologically—with the tympanitic distention resulting from oedematous infiltration of the intestinal walls, not from interruption of innervation.

The tendency to hang the panoply of medication on the peg of nomenclature has led, in turn, to the vaunting of venesection, leeching, mercurialisation, antimony, quinine, hot fomentations, cold baths or ice-bladders, as remedies for peritonitis; and to-day a therapeutic disputation weighs the opposing claims of "treatment" by opiates or by cathartics, either of which may be indicated or contra-indicated in particular cases.

In instances resulting from wounds, visceral rupture or perforation, when intensity of pain immediately menaces the heart's function, morphia is temporarily useful; but where surgical measures are so imperatively demanded, further reliance on its beneficial influence is to be deprecated, and, as in acute strangulations, few surgeons of the present day would delay operation until diffuse peritonitis declared itself. In intra-peritoneal rupture of the bladder, lives were saved by suture of the vesical rent and ablution of the abdominal cavity, in the early days of clean ventral surgery. In intussusception—granting that there are rare recoveries without peritonitis—the risk is so great that operation as soon as

impaction becomes evident is preferable to waiting for the sloughing of half a yard or so of the intussusceptum, or wasting time with ponderous doses of quicksilver or rectal inflation with the kitchen bellows. Wherever, in fine, peritoneal inflammation is manifestly caused by penetrating or strangulating local lesion, we should no more think of treatment directed to it alone than we should attempt to cure the inflammation produced by a splinter in the skin without removing the splinter. Another justification for operative intervention is the advent of purulent effusion; but here diagnosis is often at fault, since the chills and fluctuations of temperature ordinarily indicative of suppuration are not usually present. In all such surgical examples there is no longer need for the second alleged purpose of opiates, namely, to favour the formation of adhesions, or to secure their preservation by arresting peristaltic action.

But in other than surgical cases this bugbear of "peristalsis" is inconsistently dreaded; for when tympanites occurs peristalsis ceases. In peritonitis from non-perforative typhlitis, or coprostatic obstruction elsewhere, continued constipation itself increases the chance of ulceration and possible perforation; and some of those who most oppose cathartics advocate in such conditions copious and oft repeated enemata, which, if they act at all, must do so by exciting peristalsis. Small "persuasive" doses of calomel at an early stage will often happily prepare the way for slowly administered enemata, and opium, if used at all, should be limited to moderating pain. In cases of this sort, less attention than it deserves has, I think, been given to belladonna, which deadens the sensibility of the nerve-endings, and so allays pain and consequently diminishes reflex irritability, tends to relieve constipation, and supports the heart, embarrassed by pulmonary compression as well as by its own enfeeblement.

Opium—at all events, in narcotic doses—is also contra-indicated where nephritis exists; and, on account of its influence in lowering the already lessened respiratory oxygenation and retarding eliminative processes, is not rationally applicable

to cases where peritonitis is an epiphenomenon of general septicaemia, and where pain rarely calls for an anodyne. In such cases, oftenest in puerperae, foetid diarrhoea commonly coexists. The bowel is freighted with putrescent matter, and mild cathartics and intestinal antiseptics are advisable rather than pharmacological peristaltic paralysis. The same argument obtains wherever decomposition or abnormal fermentation of the intestinal contents lends added danger to peritonitis, as in non-perforative dysentery, some forms of typhlitis, and other similar conditions. The relief of meteorism by calomel (or probably by that portion of it converted into bichloride) was noted many years ago, and in the newer class of remedies, represented by salol and resorcin, we may find useful therapeutic agents in instances of this kind.

It is in "the early stage of peritonitis following operations" of abdominal section, according to Lawson Tait, that the threatened inflammation is most certainly arrested, and a fatal issue averted by "the prompt use of the turpentine enema and the Seidlitz powder;" but in view of the obscure diagnosis of this "early stage" (no well marked symptoms beyond a rise of temperature being often noticeable), I have wondered if some, at least, of these cases were not "fever of non-elimination," occasionally mistaken for septic infection. In other instances, as Dr. A. F. Currier has suggested, neglect properly to replace the omentum, or injury thereto in separating adhesions or otherwise, may be the starting-point of peritonitis; but even here I believe that the chief reliance of gynaecological operators is on saline cathartics. The further discussion of this topic, however, I shall leave to those whose experience in evisceration is greater than mine.

Where purulent exudation is present, or likely to occur, quinia, retarding leucocytic migration, is indicated; and its tonic, and possibly antiseptic, action may render it serviceable in other circumstances. In all instances where the "tendency to death" is by exhaustion, concentrated and easily assimilable nourishment, and stimulants when needed, are more valuable than drugs; and in the frequent virtual

abolition of the function of the upper part of the digestive tract, we should not forget Anstie's rectal alimentation with strong meat broths, under which pain and vomiting are sometimes allayed and the circulation strengthened.

In the uninfected forms of peritonitis, vascular derivation to the surface, by turpentine stupes and light, warm fomentations, is more philosophical than the prolonged and depressing refrigeration by ice-coils, recommended by some disciples of the German school. High pyrexia is rarely maintained long enough to require artificial abstraction of heat; on the contrary, if the issue become grave, a lowering of temperature is a common evil omen. This asthenic proclivity should also absolutely preclude cardiac depressants, such as antimony, aconite, veratrum viride, or the more mischievous group of "antipyretics" headed by the patented "antipyrene."

The clinical details of treatment in different classes of cases will surely be so thoroughly set forth by those who are to take part in the ensuing discussion, that I shall not trespass further upon your time by adverting to them. My task has been accomplished if I have sufficiently emphasised the general proposition that the therapeutic indications are not only modified by, but are altogether dependent on, the causative factors.



